"3rd—Marked asthmatic symptoms were associated with 55% of the cases. The symptoms were relieved in 84.2% of the cases."

In a personal letter to me they state: "It is a carefully standardized vaccine from the pollens of timothy, red top, June grass, orchard grass, wheat, sorrel, dock, daisy, maize, ragweed, goldenrod, all of which are known to be important factors in causing hay fever in the spring, summer and fall. Hence our vaccine offers protection against each individual pollen mentioned, as well as against all the pollens collectively."

The Mulford Company puts out two preparations, one for the spring type and one for the fall type. Their mixture corresponds botanically to the Lederle. Now compare this list of grasses and plants with the list of flora at Tonopah and Goldfields as follows:

Lederle Pollen Vaccines.	*Flora of Tonopah and Goldfield.*
Timothy,	Russian thistle,
Red top,	Red orache,
June grass,	Spiny salt bush,
Orchard grass,	Greasewood,
Wheat,	Sagebrush,
Sorrell,	Shad scale,
Dock,	Pigweed or lamb's quarters,
Daisy,	Kochia,
Maize,	Hop sage,
Ragweed,	Desert tea,
Goldenrod.	Salt grass,
	Pigweed,

and note that there is no botanical relationship between them. Nearly the same comparison can be made in other sections of the West.

Should not such commercialism be discouraged and some thought be given to develop a method which has for its main object the good of the patient? A preparation to be of value should be made with some thought of the district where it is to be sold. If not, physicians should realize at the start that no uniform results can be looked for.

The results of the work so far indicate:

1st. The value of a careful botanical survey of hay fever districts and a collection of pollens representing the principal flora of that district.

2nd. The necessity of careful biological tests with extracts of pollens from the principal flora and in some instances the use of atmospheric plates to find the unsuspected offender.

3rd. The necessity of removal of all focal infections wherever found, prior to treatment.

4th. The need of team work—i. e., the cooperation of an internist, neurologist, laboratory technician and laryngologist.

5th. That pollen therapy, to quote Hitchins, holds out promise of greater benefit to hay fever victims than any other method of treatment yet suggested.

6th. That treatment should be commenced at least sixty days before the hay fever season begins and should be continued at intervals during the hay fever period of the patient.

240 Stockton St.

McKenzie, John M.: "The Physiological and Pathological Relation Between the Nose and Sexual Apparatus of Man." Johns Hopkins Bulletin, January, 1898.
Goodale, J. L.: "Studies Regarding Anaphylactic Reactions Occurring in Horse Asthma and Allied Conditions." Transactions Laryngological Association, 1914.
Goodale, J. L.: "Pollen Therapy in Hay Fever." Transactions Laryngological Association, 1915.
Goodale, J. L.: "Diagnosis and Management of Vaso-Motor Disturbances of the Upper Air Passages." Transactions Laryngological Association, 1916.
Chappell, W. F.: "The Effects of Protein Extracts from Fruit and Pollen on the Upper Air Tract." Transactions Laryngological Association, 1915.
Brubaker, J. F.: "The Innervation of the Nasal Chambers." Transactions of the R. L. & O., 1916.
Wilson: "Some Observations on the Treatment of Hay Fever." Transactions of the R. L. & O., 1916.
Grayson, C. P.: "Pathogenesis of Nasal Neuroses." Transactions of the Laryngological Association, 1916.
Fetterolf, G.: "A Case of Vagatonia Apparently Originating in the Nasal Accessory Sinuses." Transactions of the Laryngological Association, 1916.
Oppenheim and Gotlob: "Pollen Therapy in Pollenosis." Medical Record, March, 1916.
Scheppegrell, W.: "Classification of Hay Fever Pollens from a Biological Standpoint." Boston Medical and Surgical Journal, July 12, 1917.
Scheppegrell, W.: "Hay Fever and Its Prevention." Public Health Reports, July 21, 1916.
Scheppegrell, W.: "Hay Fever." Philadelphia Medical Journal, December 9, 1916.
Scheppegrell, W.: "Cause and Prevention." American Medical Association Journal, March 4, 1916.
Scheppegrell, W.: "Its Relation to Seasons, Occupations, Sex." New York Medical Journal, December 9, 1916.
Scheppegrell, W.: "Hay Fever Pollens." Archives of Internal Medicine, June, 1917.
Scheppegrell, W.: "Ragweed Pollen in Nasal Secretions of Hay Fever Cases." Medical Record, October 4, 1916.
Scheppegrell, W.: "Cause, Treatment and Prevention of Hay Fever." Medical Record, July 15, 1916.
Scheppegrell, W.: "Hay Fever in the Southern States." Southern Medical Journal, July, 1916.
Scheppegrell, W.: "Cause and Prevention in Rocky Mountains and Pacific States." Public Health Reports, July 20, 1917.
Hitchens, Parker: "Therapeutics of Hay Fever." New York Medical Journal, October 7, 1916.
Talbot, G. H.: "Asthma in Children and Its Relation to Anaphylaxis." Boston Medical Journal, 1916.
Cook, Robt.: "Treatment of Hay Fever, Active Immunization." Laryngoscope, February, 1916.
Koessler: "Specific Treatment of Hay Fever by Active Immunization." Illinois Medical Journal, August, 1914.
Frazer, J. S.: "Nasal Neuroses, Regarded as Sensitization of Respiratory Tract." Edinburgh Medical Journal, August, 1917.
Oppenheimer & Gottlob: "Active Immunization of Hay Fever." New York Medical Journal, February, 1915.
Wodehouse: "Immuno Chemical Studies of Protein of Cat, Horse and Dog Hair." Journal of Immunology, April, 1917.
Howe: "Anaphylaxis in Respiratory Tract." New York Medical Journal, April 15, 1916.
Wodehouse: "Immuno Chemical Studies of Plant Proteins"; "Immuno Chemical Studies of Proteins." American Journal of Botany, July, 1917.
Wodehouse: "Bacteria, Food, Cat, Dog, Horse Dander." Journal of Immunology," 1916-1917.
Walker, Chandler: "Various Articles Dealing with Asthma, Associated with Proteid Anaphylaxis of Bacterial, Food and Animal Hair Proteids." Appearing in the Journal of Medical Research. Journal of Medical Research, Vol. XXV, 1916-1917.

THE COLLOIDAL GOLD (LANGE) TEST IN DIAGNOSIS. U. C. HOSPITAL.

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The readiness with which cerebrospinal fluid is obtained by lumbar puncture has led in recent years to a study of diagnostic methods applied to it. No study of disease of the cerebrospinal nervous system is complete without the valuable evidence derived from the application of the various tests to the cerebrospinal fluid, such as: the Wassermann test, the Noguchi butyric acid test, the Nonne test for globulin, the Fehling's reduction test, and the cell count. In 1912, Lange added to these the colloidal gold test. It is the purpose of this paper to record the application of this test to about one hundred fluids, to show its value as a diagnostic method in diseases of the central nervous system and to emphasize the im-

portance of applying it more generally in the study of our cases.

If to a definite quantity of colloidal gold solution a given quantity of sodium chloride solution be added, particles of colloidal gold slowly precipitate out, leaving a clear solution. Zsigmondy discovered that albuminous bodies prevent this precipitation. If, for example, a definite quantity of colloidal gold solution is placed in a beaker with a given quantity of an electrolyte such as sodium chloride, and a known amount of an albumin added, the colloidal gold solution will remain unchanged; in other words, the albumin has inhibited the precipitating action of the electrolyte on the colloidal solution. Zsigmondy further showed that of each albumin a quantity different from every other albumin is required to prevent the precipitation of the same amount of colloidal gold in a sodium chloride solution. This fact serves to identify the albumin, and it is possible to determine by the gold chloride reaction what albumin is present and in what amount. In the cerebrospinal fluid various albumins are present, and Lange attempted to study them by the application of this method. This is what he found:

A normal cerebrospinal fluid does not precipitate a colloidal gold solution in sodium chloride.

Syphilitic fluids produce a characteristic reaction which is different in paretic cases on the one hand and tabetic and cerebrospinal syphilitic cases on the other.

Fluids from suppurative cases and in brain tumor, hemorrhage, and other non-syphilitic conditions likewise produce characteristic reactions, but different from those in syphilitic fluids.

These facts are of great importance in the diagnosis of cerebrospinal disease. Without the reaction it has been possible by a study of the cells and the Wassermann test to differentiate between specific and non-specific disease of the cerebrospinal system. With the reaction, besides affording substantiating evidence for the interpretation of the other tests, it has become possible to differentiate cases of paresis from cerebrospinal lues and tabes, as well as to distinguish between specific and non-specific central nervous system disease.

In 1913, Sippy and Moody investigated the Lange reaction in the spinal fluid from 268 cases, using the Nonne test for globulin as a control. They strongly advocated its application in cases of cerebrospinal disease. Grulee and Moody at the same time reported nine cases clinically congenital lues and seven suspected cases, and showed that the Lange reaction confirmed cases with clinical signs of congenital syphilis. Lee and Hinton concluded that the test is more delicate than the blood Wassermann, fluid Wassermann, cell count, and globulin, and that it is typical for syphilis of the central nervous system. They believed it possesses an advantage in that it gives a reaction with pathological fluids other than syphilitic fluids; and they considered the margin of error in performing the test small. Weston, Darling and Newcomb found in its application to psychiatry a very valuable adjunct to the prevailing clinical tests. They found that in cases of clinical cerebrospinal lues the fluid Wassermann was always positive and the gold test always positive. In paretic fluids the gold test was always positive. In dementia praecox, manic-depressive insanity, arteriosclerotic dementia, and epilepsy there was no typical reaction. In unclassified psychoses the fluid Wassermann and gold test ran a parallel course. Miller and Levy, and later Miller with several collaborators, have made a careful study of the reaction to determine, if possible, methods of eradicating the inconstancy of the results they found they had been getting. They observed that the gold test may be positive in cases in which all other pathological findings are absent, that it may occur in cases not clinically paresis, that in general paresis the absence of dementia does not argue against the value of the test, and that in congenital lues, except in juvenile paresis, the test should not be relied upon to the exclusion of other laboratory tests. They affirm that it does not replace other tests, but offers very valuable confirmatory evidence. Others in Europe and in this country have investigated the test in its application to spinal fluid. In general it is the opinion that the reaction is of great value in the hands of careful workers; but one and all agree that, while the test itself is simple, the preparation of a satisfactory indicator is extremely difficult and is practically the only bar to a more general application of the colloidal gold reaction.

In preparing the indicator the following reagents are used: a 1% solution of gold chloride; a 2% solution of potassium carbonate; a 1% solution of formalin. The chemicals must be chemically pure and the solutions must be made in distilled water. To prepare half a liter of indicator, 500 c. c. of freshly distilled water are placed in a beaker of Jena glass and slowly heated to 60° C. At this point 5 c. c. of gold chloride solution and 5 c. c. of potassium carbonate solution are quickly added to the water with a sterile pipette and the gas then turned on full until the solution attains a temperature of 95° C. The heat is now removed and 5 c. c. formalin solution added slowly with constant stirring. The solution which has remained colorless until the addition of the formalin now turns pink, then lilac, then light red, and finally deep red with just a glint of orange. The indicator thus formed should be clearly transparent by direct or reflected light. If it is purplish and smoky it is unsatisfactory and the attempt to prepare it should be repeated, there being no possible way of correcting it once the reduction has taken place. Experience has shown that only when the directions for preparing the indicator are implicitly followed may one expect to obtain satisfactory results. The glassware must be cleansed chemically, and the water used must be doubly or even triply distilled with an apparatus free from rubber connections. Such an apparently insignificant substitution as the use of a flask for a beaker in the preparation of the indicator may lead to a failure. If the solution is heated too rapidly failure will likewise result. Instead of formalin, glucose has been used as a reducing agent and Miller reduces with formalin

after the addition of a few drops of oxalic acid; but I have found the original method of Lange to give the best results if it be followed with the utmost care. It is obvious that the successful preparation of the indicator depends on the care of a skillful laboratory worker. Once the indicator is prepared the carrying out of the test is a very simple matter.

Eleven chemically clean tubes are set up and 1 c. c. of .4% sodium chloride added to each. To the first tube is added an additional .8 c. c. of salt solution and .2 c. c. of the spinal fluid to be tested. One c. c. is now removed from the first tube and placed in the second. By the successive removal of 1 c. c. from each tube to the next, dilutions of the fluid are prepared in geometrical progression from 1:10, 1:20, 1:40, etc., to 1:5120. The eleventh tube, containing only salt solution, is used as a control. To each tube is now added 5 c. c. of the indicator, and the tubes are set aside for eighteen hours.

The changes which take place in the tubes are most interesting. The reaction, we must remember, depends upon the prevention by the albumin in the spinal fluid of the precipitation of the colloidal gold in the presence of the electrolyte, sodium chloride. In the absence of these proteins precipitation takes place in varying amounts, depending on the dilution, so that the indicator assumes different colors in the different dilutions, by the clumping together of the small colloidal particles. The color changes vary from deep red to red-blue, to lilac or purple, to blue, to cloudy, to complete precipitation resulting in a clear tube. For convenience of expression in reporting the results of a test the changes are numbered from 0 to 5, 0 being the normal color of the indicator and 5 clear.

Having described with all possible brevity the preparation of the indicator and the performance of the test, let us study its application to about 100 fluids obtained in the examination of patients in the wards and Neurological Clinic of the University of California Hospital. To these cases are added a few obtained from private sources.

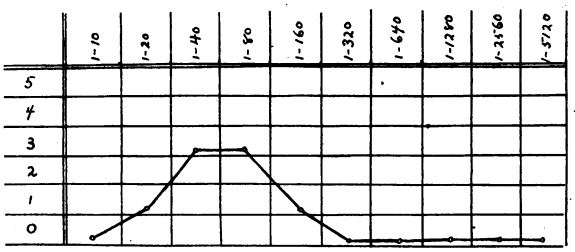

Chart I – Luetic Curve.

Chart I represents a typical curve constructed with the abscissae representing the different dilutions, and the ordinates the varying color changes. The apex is attained at dilutions 1:40 and 1:80 with a color change to blue. At the dilutions 1:320 the curve becomes coincident with the base line. This is a typical "luetic curve," found in cases of tabes, cerebrospinal lues, and congenital lues.

A normal fluid would be expressed by a straight line through all dilutions coincident with the base-line, there being no clumping of colloidal gold in the normal case.

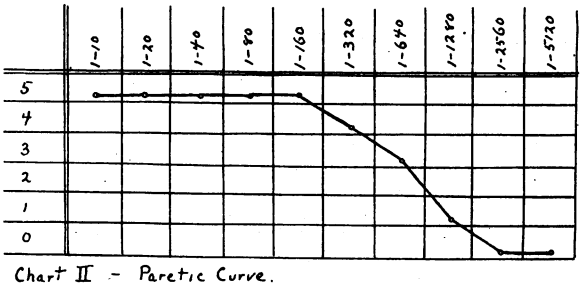

Chart II – Paretic Curve.

Chart II represents a typical curve obtained in a case of general paresis. Complete precipitation occurs in the low dilutions to a dilution of 1:160. In the higher dilutions varying degrees of inhibition result in successive color changes until the color of the indicator is reached at a dilution 1:2560. Comparing Charts I and II we see at a glance the possibilities of the test in differentiating the two conditions of which they are the graphical representations.

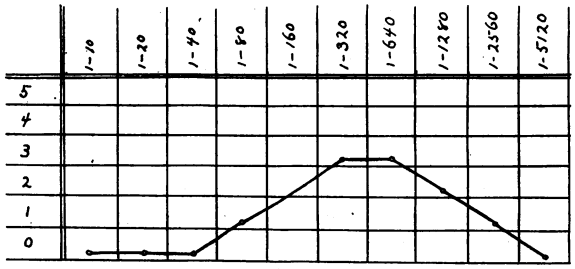

Chart III – Non-luetic Curve.

Chart III is a curve resulting in a case of non-syphilitic disease of the central nervous system. The apex is attained in the higher dilutions 1:320 and 1:640. Such a curve would be typical, for example, of tubercular meningitis.

An analysis of each of the cases studied would be very instructive, but for conciseness the records have been grouped so as to show the conformity of the color changes in each group with the type of reaction. The blood test and spinal fluid findings are likewise grouped so as to show a parallel with the colloidal gold reaction.

Table I shows the gold chloride dilutions giving the strongest precipitation in the different groups of cases, together with the average cell count for each group, the number of positive and negative Nonne and Noguchi tests, and the number of positive and negative blood and fluid Wassermann tests.

Summary. Table II. In seven cases which were clinically general paresis every case showed a paretic curve in the colloidal gold test, while only five gave the globulin and Wassermann tests positive throughout. The value of the Lange in diagnosis as shown in this group is further illustrated in the case of juvenile paresis. This case, clinically a congenital lues, with few neurological signs and no mental changes, has been observed in hospital over a period of several weeks during which time intensive treatment has been adminis-

TABLE I.

Diagnosis	No. of Cases	Strongest Color Change	Cells per 1 c.m.m.	Noguchi +	Noguchi −	Nonne +	Nonne −	Wassermann Blood +	Wassermann Blood −	Wassermann Fluid +	Wassermann Fluid −
General Paresis	7	1:10-1:640	Av. 45	6	1	5	2	5	2	5	2
Tabes	9	1:80-1:320	Av. 17	4	5	6	3	3	6	5	4
C. S. Lues	22	1:80-1:320	Av. 56	17	5	18	4	12	10	14	8
Syph. Meningitis	2	1:80-1:640	Av. 425	2		2		2		2	
Congenital Lues	3	0	Av. 3		3	1	2	3			3
Juvenile Paresis	1	1:10-1:640	Av. 16	1			1	1		1	
Latent Syphilis	1	1:80	1	1		1			1		1
Epilepsy	3	0	Av. 4	1	2	1	2		3		3
Malignant Lues	1	0	8		1		1	1			1
Secondary Lues	9	0	Av. 5	5	4	3	6	5	4	2	7
Paralysis Agitans	1	0	2		1		1		1		1
Typhoid	1	0	0		1		1		1		1
Malaria	1	1:160	7		1		1		1		1
Delirium Tremens	1	0	2		1		1		1		1
Dementia Praecox	1	0	8		1	1			1		1
Miscellaneous	16	0	Av. 6	5	11	6	10		16		16

TABLE II.

Diagnosis	No. of Cases	Positive Colloidal Gold	Positive Blood Wassermann	Positive Fluid Wassermann
General Paresis	7	7	5	5
Juvenile Paresis	1	1	1	1
Tabes	9	8	3	5
C. S. Lues	22	16	10	14
Congenital Lues	3	Negative 3	3	Negative 3
Miscellaneous	43	Negative 43	Negative 43	Negative 43
Secondary Lues	9	Negative 9	5	2

tered. The spinal fluid has been examined six times. Each examination shows a diminished cell count and a diminution in globulin and in the positive blood Wassermann; but the fluid Wassermann remains double positive in a dilution of 0.5 and the colloidal gold test is practically unchanged from what it was in the beginning. That the case is progressing in spite of treatment is evident from the pupillary changes. In prognosis, therefore, the test assists us definitely and is to be given incomparably more weight than a study of the successive cell counts and globulin reactions. Another case, clinically cerebral lues, with positive cerebrospinal fluid findings, showed a colloidal gold test giving a paretic curve. Several weeks later mental symptoms, such as loss of memory and depression, have begun to appear and we feel justified in anticipating the development of paresis. Another case gives an almost identical course, with a positive paretic curve.

In nine cases which were clinically tabes every case but one gave a luetic curve in the colloidal gold test. In contrast with this result, only about one-half gave positive specific findings in the blood and fluid examinations. The one case which did not show a luetic curve had received Swift-Ellis intraspinal treatment, KI, and mercury over a period of more than a year.

In 22 cases of cerebrospinal lues 16 showed a luetic curve while the Nonne globulin test was positive in 18, the blood Wassermann positive in 10, and the fluid Wassermann in 14. The luetic curve, therefore, is confirmatory of other fluid findings in most cases of tabes and cerebrospinal syphilis and is positive oftener than the other spinal fluid tests.

In three cases of congenital lues without neurological symptoms and signs, the colloidal gold test was negative and parallel with the other spinal fluid findings.

In 23 cases with negative spinal fluid tests the colloidal gold reaction was likewise negative.

In nine cases with secondary syphilis, five showed increased globulin and two a positive Wassermann in the fluid, but the colloidal gold test was negative in all.

In one case diagnosed latent syphilis the spinal fluid findings were negative except for a positive Noguchi and a positive colloidal gold test.

Other miscellaneous cases to the number of about twenty were negative throughout.

Several fluids examined from cases of cerebral hemorrhage occurring months previously showed no characteristic gold curve. No case of tubercular or epidemic meningitis or of brain tumor is included in the series, but the testimony of others is strongly in advocacy of the colloidal gold test in these conditions.

Conclusions. Observations on this series support the opinion of previous workers, that while the colloidal gold test is valuable, it does not replace other tests but confirms them and in some instances assists in a prognosis. The test is valueless unless a satisfactory indicator is prepared. It is simple of execution, and the error is small if the precaution be observed of obtaining blood-free spinal fluid in clean, sterile tubes. In congenital lues the reaction does not add to the evidence given by other spinal fluid tests but it is of confirmatory value. In tabes the test, besides confirming evidence from other sources, may, when it gives a paretic curve, predict the development of a paresis. In tabes and cerebrospinal lues it may be positive in cases in which the Wassermann, cell count, and globulin are negative. In general paresis it is invariably positive and is of absolute value in differentiating between general paresis on the one hand and tabes and cerebrospinal lues on the other. In normal fluids it is invariably negative, if Miller's rule of counting all color changes below 2 as negative be followed. Where the laboratory facilities are such that care and time may be devoted to the preparation of a suitable indicator the test should be performed on every spinal fluid; the data for diagnosis is incomplete otherwise. In these days of scientific methods in medicine, when we are aiming at precision in diagnosis, I wish to advocate strongly this test,

at once simple and precise, in aiding in correct diagnosis and prognosis.

Bibliography.

Zsigmondy: Zeitsch. f. Chemotherapie, 1912, 1, 44.
Lange: Berl. klin. Wochenschr., 1912, xlix, 897.
Sippy and Moody: Trans. Assn. Am. Phys., 1913, xxviii, 720.
Grulee and Moody: Jour. Amer. Med. Assn., July, 1913, 13.
Lee and Hinton: Amer. Jour. Med. Sci., 1914, cxlviii, 33.
Weston, Darling and Newcomb: Amer. Jour. Insan., 1915, lxxi, 773.
Miller and Levy: Bull. Johns Hop. Hosp., 1914, xxv, 133.
Miller and Brush: Bull. Johns Hop. Hosp., 1915, xxvi, 391.

Discussion.

Dr. E. A. Victors: There have been a number of objections to the colloidal gold reaction on account of the difficulty of preparing a proper reagent and that gold is one of the most unstable of colloids. In certain of the larger institutions where the reaction can be properly controlled and where fluids can be properly taken, it is a test of much value. The fluid should be taken with a properly prepared platinum needle and must be absolutely free from blood. I do not advise the colloidal Lange test as an ordinary laboratory procedure. Its quantitative or diagnostic factors are of importance although at times differentiation between tubercular meningitis and specific cerebrospinal disease can not be made.

Recently Emmanuel has suggested a colloidal mastic test. This is a far more stable colloid and exceedingly simple to prepare and the errors that creep into the gold test do not exist here. I am presenting at a coming meeting several hundred of these tests. The parallel between the mastic asd Wassermann reaction is remarkably striking and it seems every bit as delicate as the colloidal gold, although not as differential.

Dr. Hans Lisser: I think Dr. Harvey's paper deserves considerable discussion, for the conclusions are well worth while. A diagnosis of paresis involves a prognosis so portentous that any test which will permit of our recognizing this disorder earlier than we can do so clinically, while there may still be a possibility of intensive prophylactic treatment, is exceedingly valuable. Whether the Lange test will accomplish this much desired result remains to be seen; but in any case we should be very sure that the reaction is done by competent hands.

Dr. Harvey mentioned one case that interested us very much—probably a case of juvenile paresis. A boy, seven years old, came to the children's department of Dr. Lucas, at the University of California Hospital, and a Wassermann in the blood was done because he had markedly unequal pupils. One pupil was very much dilated and did not react to light at all. The other pupil reacted to light when we first saw him, not very actively, but definitely. The Wassermann in the blood was XX, on the basis of XXX being strongly positive. His spinal fluid showed 54 cells, positive Nonne, questionable Noguchi and XXX positive Wassermann in .2 cc.

Here was a patient with an excellent mentality of seven years plus; emotionally normal, showing no psychic evidences of paresis. Moreover, the neurological examination showed no signs of involvement, except for the eye findings just mentioned. The Lange gold test, however, gave a marked reaction in the paretic zone. I might add too, making the case still more peculiar, that this boy showed no evidence of optic atrophy, which is so frequently present in juvenile paresis.

We began to treat him with intravenous salvarsan, iodides and mercurial inunctions, in order to observe what results could be obtained before resorting to intraspinous therapy. Parenthetically it may be remarked, that had we given intraspinous therapy at once, the changes that occurred would have been ascribed to the "wonders" of intradural treatment, though in this case these questionable "wonders" were accomplished by more simple methods. This seven-year-old boy received, first—.45 Neosalvarsan, then .6; then .9; the full adult dose. Since that time he has had four more full doses of .9 neosalvarsan. We are not recommending such enormous doses, but it is interesting to place them on record. He vomited somewhat after the larger doses, but hardly more so than many adults.

Now for the results—the cells in the spinal fluid decreased from 54 to 3, Nonne and Noguchi disappeared, the Wassermann became negative in .2, questionable in .3 cc. His mentality remains normal. The pupil which was fairly active in the beginning is now barely reacting to light. Throughout all this treatment the colloidal gold curve remained strongly paretic.

Accordingly on the basis of one laboratory test alone, we are making a diagnosis of juvenile paresis, even though there are no clinical signs to substantiate it. It will be interesting to see what happens to this boy in the next two or three years.

Dr. H. G. Mehrtens: Dr. Harvey did not mention one of the things that arise as a confusing factor in the Lange test. If a patient has had previous intradural treatment, that has an effect on the fluid as far as the Lange test is concerned. In the neurological clinic at Stanford we followed this rather carefully and found some very odd results. In certain cases the Lange test is reduced steadily, finally becoming negative in the last of the reactions. But frequently, especially in paresis, the Lange test does not become negative, but becomes confused, takes on odd and atypical forms, so that if the patient should arrive and should not disclose the history of former treatment, and a lumbar puncture were done, the test would be of little or no significance. We had a case in point, in which the patient intentionally withheld the information of previous intradural treatment. The signs were perfectly evident, and a diagnosis of probable paresis was made. The Lange came back with a very atypical form, so much so it was not even typical for tabes, cerebrospinal syphilis or meningitis. Later on the patient volunteered the information that intradural treatments by both the Byrnes and Swift Ellis methods had been given, explaining the peculiar form of the Lange test.

Dr. Harvey, closing discussion: The observation of Dr. Mehrtens is certainly interesting. We had occasion to notice one case of tabes that had received several treatments of Swift-Ellis, and in which the Lange test was negative.

Dr. Lisser did not mention that in the case of general paresis, the mastic test was positive, corroborating the colloidal gold. We have been using the mastic test for some months at the University and are glad to know that a report on it will be made here.

WHY OPTICIANS OR OPTOMETRISTS OUGHT TO HAVE MEDICAL DEGREE.

(a) A general knowledge of the entire human body is necessary for the intelligent treatment of eye diseases.

(b) The majority of eye diseases are not to be cured by the mere fitting of glasses.

(c) Systematic and medical treatment is required in 70 to 90 per cent. of all eye diseases.

(d) Many diseases have their first or most diagnostic signs appear in the eye.—Bull. of Fed. of State Med. Bds., Feb., 1918.